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The effects of pempidine and hexamethonium on release of antidiuretic hormone by nicotine and osmotic stimuli in the cat

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The synapses at the supraoptic nuclei are thought to be cholinergic (Pickford, 1939, 1947) and pharmacologically may resemble autonomic ganglia (Walker, 1957). Bisset & Walker (1957) showed in rats that nicotine releases both antidiuretic hormone (ADH) and oxytocin and release is not diminished by hexamethonium. In contrast reflex release of oxytocin to suckling is abolished by pentolinium (Chaudhury, 1961).

In these experiments using cats anaesthetized with chloralose, 4 ml. blood samples were collected 5 min before and 2 and 40 min after intracarotid injection of nicotine hydrogen tartrate (40 μg nicotine base/kg) or 1 ml. M sodium chloride solution. Following intravenous pempidine tartrate (5 mg/kg) or hexamethonium bromide (5 mg/kg) the stimulus was repeated and blood samples taken as before. The blood was extracted according to the method of Bisset, Hilton & Poisner (1967) and assayed for antidiuretic activity on alcohol anaesthetized rats (Bisset, 1962).

Experiments showed that nicotine released ADH even when carotid chemoreceptors had been denervated. The results summarized in Table 1 demonstrate that the release of ADH by nicotine is prevented by pempidine but not by hexamethonium. In contrast, pempidine does not block release of ADH by osmotic stimulation. Table 1 also shows that the concentration of ADH in the blood increased after pempidine but not after hexamethonium.

The effect of pempidine on ADH release by nicotine may result from blockade of central synapses which are not reached by hexamethonium. The failure of

TABLE 1. *Effects of intravenous pempidine* (5 *mg/kg*) (*P*) *and hexamethonium* (5 *mg/kg*) (C_6) *on release of ADH by intracarotid nicotine* (40 μg/*kg*) (*N*) *and sodium chloride solution* (1 *ml.* M) *NaCl*

Time of blood sample (min)	ADH (μ-u./ml. blood)							
	−5	0 Stimulus	+2	+40	Blocking agent	−5	0 Stimulus	+2	+40
		↓N					↓N		
	<5		18	7·5	P	22		12·5	<5
	5		18·5	8·5	P	15		3·5	<2
	<2		5	2	P	11		10	<2
	<5		8	<5	P	400		15	<5
		↓N					↓N		
	9·5		30	15	C_6	34		48	20
	<3		7	3·5	C_6	3		7·4	4
	17		34	7	C_6	6		17	11
		↓NaCl					↓NaC		
	9·4		25	9·4	P	31		50	12·5
	12·5		45	12·5	P	100		400	100
	6		36	16	P	23		100	90

pempidine to block ADH release by osmotic stimulation suggests that this stimulus may not involve a cholinergic link. The increase in circulating ADH following pempidine may be due to blockade of inhibitory pathways.

This work was supported by a grant from the Medical Research Council.

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The effect of *p*-methoxyphenylethylamine (PMPEA) on monosynaptic reflexes in the cat

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The actions of PMPEA and several blocking agents were studied in cats anaesthetized with chloralose. Flexor (posterior biceps, semitendinosus, PBST) and extensor (gastrocnemius, soleus, GS) monosynaptic reflexes were recorded from peripheral nerves of the cat hindlimb in response to stimulation of dorsal roots of the lumbosacral enlargement. The L_6–S_1 dorsal roots were sectioned bilaterally, and the spinal cord was transected at L_1. Monosynaptic reflex spikes were integrated electronically. The integrals were displayed, along with a record of the arterial blood pressure, on a pen-recorder. The drugs used included, in addition to PMPEA, phenoxybenzamine, methysergide and pronethalol; administration was intravenous.

PMPEA (5 mg/kg) increased the monosynaptic reflexes of both flexor and extensor motoneurones. The potentiation did not exhibit tachyphylaxis to repeated doses of the compound. The effect of pretreatment with phenoxybenzamine (20 mg/kg), methysergide (2 mg/kg) or pronethalol (5 mg/kg) was investigated. Pretreatment with either phenoxybenzamine or methysergide reduced the response to PMPEA. The presence of both phenoxybenzamine and methysergide produced almost complete block of the PMPEA response. Pretreatment with pronethalol had little effect.

The possibility that 5-hydroxytryptamine and noradrenaline have excitatory effects on monosynaptic reflexes in the cat cord has been postulated (Baker & Anderson, 1965; Anderson & Shibuya, 1966). These authors demonstrated that pretreatment with 5-hydroxytryptophan, L-tryptophan or 1-3,4,dihydroxyphenylalanine increased the size of the monosynaptic reflex recorded from the cat cord. The site of action of PMPEA may be on 5-hydroxytryptamine and/or catecholamine receptors in the spinal cord, although the possibility of release of monoamines within the spinal cord requires further investigation.